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ABSTRACT

Alcoholics Anonymous was founded in 1935 and observed its 50th anniversary in 1985. This document discusses the changes that have taken place in knowledge about alcoholism. Specifically, this booklet compares what behavioral scientists knew about alcohol abuse and alcoholism in 1935 and what they knew and could do about them in 1985. Contributions were chosen that meet these criteria: they are of clinical as well as scientific significance; they were the object of serious scientific study; and behavioral scientists have been extensively involved in these fields of enquiry. The four areas of focus include: behavioral pathology, etiology, treatment, and prevention. In the area of behavioral pathology, the theories of behavioral tolerance, craving and loss of control, alcoholism and moral insanity, alcoholism and depression, and alcoholism as a disorder of man are discussed. Theories of etiology in 1935 and 1985 are compared. Treatment regimes and prevention approaches in 1935 and 1985 are also compared. The report concludes that significant progress has been made in the past 50 years on identifying and understanding important effects of alcohol on human behavior, but progress in research on factors other than genetic predisposition has been limited by the expense, complexity, and difficulty of longitudinal research. (ABL)

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About the Author

Born and raised in Saint Louis, Missouri, Peter E. Nathan received the A.B. in social relations with honors from Harvard College in 1957 and the Ph.D. in clinical psychology from Washington University in 1962. During seven years on the faculty of Harvard Medical School from 1962 to 1969, most of them spent at Boston City Hospital, Dr. Nathan launched a program of research on basic psychosocial variables associated with alcoholism that continues to the present day.

On moving to Rutgers, the State University of New Jersey, in 1969 to become Professor and Director of Clinical Psychology Training in the Department of Psychology, Dr. Nathan founded the Alcohol Behavior Research Laboratory, which continues to thrive at Rutgers. In 1974 Nathan was also named as Chair of the Department of Clinical Psychology at Rutgers' Graduate School of Applied & Professional Psychology. In 1983, he was named Henry & Anna Starr Professor of Psychology and appointed Director of Rutgers' Center of Alcohol Studies. In 1987, he began a partial leave from Rutgers to serve as Senior Program Officer for the MacArthur Foundation in Chicago.

Dr. Nathan has served as member, then chair, of the NIAAA's Alcoholism and Alcohol Problems Review Committee, member of NIMH's Psychological Sciences Fellowship Review Committee, and member of the VA's Behavioral Sciences Research Evaluation Committee. He has written or edited 10 books and well over 150 journal articles and book chapters, the majority of them detailing basic behavioral phenomena associated with alcohol abuse and dependence. He has served on many journal editorial boards, including eight years as Associate Editor of the *American Psychologist*. He currently serves as Executive Editor of the *Journal of Studies on Alcohol*. Dr. Nathan is a Past President of the American Psychological Association's Division of Clinical Psychology.

What Behavioral Scientists Know — and What They Can Do — about Alcoholism

by

Peter E. Nathan, Ph.D.

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What Behavioral Scientists Know — and What They Can Do — about Alcoholism

The fellowship of Alcoholics Anonymous was founded a bit more than 50 years ago, in 1935, in Toledo, Ohio. In my reflections on the topic of this contribution to this pamphlet series, AA's special anniversary has had an impact. What changes in what we know about alcoholism have taken place, I began to wonder, during the years separating the founding of AA and now? How much more do behavioral scientists know now about the effects of alcohol on behavior? What have we learned of etiology? Are we more effective in our treatment efforts? And can we more readily prevent alcoholism than before?

Not only was 1985 the golden anniversary of Alcoholics Anonymous, the year also coincided with a like anniversary of the formal beginning of the Center of Alcohol Studies at Yale; this research institution, now at Rutgers, has lent great scientific respectability to the enterprise of alcoholism research. Not long after the Center's founding, some of the faculty of the Center of Alcohol Studies launched the *Quarterly Journal of Studies on Alcohol*, then and now the premier journal in the field. Many alcoholism workers date the beginnings of serious efforts to treat alcoholism to the founding of Alcoholics Anonymous, and the beginnings of concerted scientific efforts to understand the actions of alcohol and the antecedents and consequences of alcoholism to the creation of the Center of Alcohol Studies and the launching of the *Journal*. Accordingly, 1985 seemed a fine year to look back to assess progress made in understanding, treating, and preventing alcoholism over the span of 50 years.

It is impossible to chronicle all advances in the varied disciplines that have contributed to knowledge about alcohol, alcoholics, and alcoholism. Instead I confine my efforts here to two sufficiently difficult tasks: Determining, first, what behavioral scientists knew about alcohol abuse and alcoholism and what they could do about them in 1935, and second, what they know and can do now, in the mid-1980's. I have chosen to focus on contributions that meet three criteria: *1.* They are of clinical as well as scientific significance; *2.* They are the object of serious scientific study; and *3.* Behavioral scientists have been involved extensively in these fields of enquiry. The four areas of focus include: *behavioral pathology, etiology, treatment,* and *prevention.*

BEHAVIORAL PATHOLOGY

Behavioral Tolerance

Behavioral Tolerance — 1935. The phenomenon of behavioral tolerance to alcohol was well recognized in 1935. Tolerance refers to the curious fact that heavy drinkers, among them alcoholics, can usually drink more alcohol than lighter drinkers. They can usually continue to function at blood alcohol levels that would disable others and, at times, actually seem to function better when they are intoxicated than when they are sober. All of these are hallmarks of behavioral tolerance, a common consequence of heavy drinking and alcoholism.

In 1935 and most of the following three decades, this behavioral tolerance was presumed to reflect two factors on which alcoholics and nonalcoholics differed: A history that had provided more than enough experience in learning to compensate for alcohol-induced behavioral impairment, and a familial and cultural milieu which provided equally extensive experience with parents and others who often drank substantial quantities of alcohol but did not always seem impaired. These causal attributions of tolerance, nonempirically derived for the most part, reflected a prevailing societal view that alcoholics tend to beget alcoholics by perpetuating for their children a morally- and culturally-deviant lifestyle. These attributions were associated with a surprising paucity of empirical research on tolerance formation and maintenance in human beings that has lasted virtually to this day (Jellinek and Jolliffe, 1940; Jellinek and McFarland, 1940; Tabakoff and Rothstein, 1983).

Some commentators explained tolerance by noting their conviction that alcohol affects alcoholics differently than it affects nonalcoholics; this view became more influential following AA's advocacy of what came to be called the "disease model of alcoholism." Nonetheless, the relevance of differences between reactions to alcohol in understanding tolerance remained unclear. Said another way, even in the face of the growing influence of the disease model, tolerance continued to be viewed by many as primarily a function of cultural and personal experience with alcohol rather than as a pharmacologic phenomenon.

In reviewing the 1930's literature on tolerance, I was surprised to note that both the ubiquity and potential role of tolerance in the diagnosis of alcoholism were generally unacknowledged by other than a few pharmacologists (e.g., Mirsky et al., 1941; Newman, 1940): Those who diagnosed and treated alcoholics rarely had much to say about a phenomenon that vastly complicated their treatment efforts yet could also have aided their diagnostic efforts.

Behavioral Tolerance — 1985. Much more about behavioral tolerance to alcohol has been studied, written about, and understood in

the decade of the 1980's than in the decade of the 1930's. It is now recognized by most biobehavioral scientists, for example, that, as people drink more, they actually increase their capacity to metabolize ethanol. The most common mechanism involves an increase in the rate at which heavy drinkers produce the alcohol-metabolizing enzyme called alcohol dehydrogenase (Li, 1983). In other words, a biophysiological explanation based on experimental evidence has been added to the earlier view that tolerance is a function of learning and environment.

As well, after prolonged and very heavy drinking, perhaps of a kind found only in alcoholics, persons may develop an additional pathway for the metabolism of alcohol, called the microsomal ethanol oxidizing system (MEOS), a membrane-associated system. Some researchers (e.g., Lieber, 1980) have claimed that the MEOS can account for as much as 20 to 25% of the ethanol oxidation rate of the liver, while others consider its contribution to be insignificant (e.g., Berry et al., 1980).

Of greater interest to behavioral scientists are reports in the past decade which suggest that ethanol and drug tolerance effects are also influenced importantly by Pavlovian conditioning phenomena. Research by Siegel (1975) on tolerance to morphine in rats led him to hypothesize that cues routinely paired with morphine become conditioned stimuli eliciting a conditioned response that tends to be opposed in direction (antagonistic) to the direct effects of morphine. With repeated pairings of environmental cues and drug, the conditioned response becomes stronger, leading to a decrease in drug effects. Tiffany and Baker (1981), among others, have confirmed Siegel's initial demonstration in rats, and Crowell et al. (1981) have done so for ethanol tolerance. Recently, Shapiro and Nathan (1986) reported data indicating that human drinkers also learn to anticipate the effects of ethanol on behavior and, via a Pavlovian conditioning mechanism, demonstrate the compensating behaviors which we call tolerance.

Craving and Loss of Control

Craving and Loss of Control — 1935. It was widely believed, even before the decades of the 1930's, 1940's, and 1950's, that alcoholics will always choose to drink as much alcohol as they can for as long as they can. This belief (still widely accepted) reflected the assumption that loss of control is so strong and so invariant that it is a central component of the behavior of all drinking alcoholics. This view was reinforced by the strong advocacy for the disease model of alcoholism the AA philosophy represents. A correlative assumption is that every alcoholic craves alcohol when sober and that it is this craving which leads the alcoholic to return to alcohol after a period of sobriety. It was assumed in those years that the alcoholic's personality, interacting with a "debauched" lifestyle, was unable to postpone

gratification or to do without pleasure (Fleeson and Gildea, 1942; Lewis, 1940). The advent of AA added to this explanation the conviction that alcoholics were burdened, as well, by a unique biophysiological mechanism — by some sort of unidentified lesion in the central nervous system — responsible for these two hallmarks of the disease model of alcoholism.

Craving and Loss of Control — 1985. A great deal of research on craving and loss of control, most of it by behavioral scientists, has taken place during the decades of the 1960's, 1970's and 1980's. This research has revealed the following:

1. Contrary to the view still current among some of those who support the disease model of alcoholism, convincing data to the effect that some alcoholics moderate their drinking have been reported since the 1960's. Alcoholics have demonstrated this capacity in the experimental laboratory, when reinforcing stimuli support either termination or moderation of drinking for varying periods of time (Bigelow et al., 1972; Mendelson and Mello, 1966; Nathan et al., 1970), as well as in the natural environment, where it seems that many alcoholics move among periods of controlled consumption, abstinence, and abusive drinking (Davies, 1962; Pattison et al., 1977).

2. The data gathered on craving, like the data on loss of control, differ from the views held by some supporters of the disease model of alcoholism and from widespread general belief: Craving for alcohol is not necessarily either a consistent or the most important threat to sobriety for the recovering alcoholic. Research on expectancies indicates instead that craving may be largely a function of an alcoholic's belief that a "priming dose" of alcohol has been consumed and that, accordingly, craving will be experienced psychologically, rather than because of a pharmacologic action of alcohol (Cutter et al., 1970; Marlatt et al., 1973).

   Relevant to this issue, as well, is the work of Marlatt and his colleagues on relapse. To them, the likelihood of relapse is far greater if the sober alcoholic anticipates the inevitability of craving and loss of control and holds to the abstinence violation effect, i.e., the belief that a single drink will invariably break abstinence and lead to uncontrolled, abusive drinking. Marlatt and his colleagues write that it is necessary to help the recovering alcoholic to understand the phenomena of relapse as they relate to recovery, to view a single "slip" as a very unpleasant but not necessarily disastrous signpost on the road to recovery, and to develop skills to cope with the phenomena associated with relapse. If this is done, the recovering alcoholic will be more likely to put craving and loss of control into context and

will feel more secure so that a drink or two will not lead to a return of abusive drinking (Marlatt, 1978; Marlatt and Gordon, 1985).

3. Craving and loss of control, hallmarks of the disease model of alcoholism, were viewed earlier as consequences of an actual physical lesion of some sort. In the face of data pointing strongly to the role of environmental and expectancy factors in the behavior of the alcoholic, craving and loss of control as well as the disease model itself have come to be regarded by many as metaphors rather than as literal representations of the state of affairs affecting these constructs. That is, the model can be reconciled with existing empirical data if the cause of craving and loss of control is seen as either a physical or a psychological lesion internal to the organism that is also affected by environmental and cognitive factors.

Alcoholism and Moral Insanity

Alcoholism and Moral Insanity — 1935. Throughout the centuries it was widely believed, first, that alcoholism leads to antisocial and psychopathic behavior, i.e., moral insanity; and second, that moral insanity causes alcoholism. In its extreme, this remnant of the moralistic beliefs of temperance workers also required the conviction both that poverty, immorality, ignorance, and atheism cause alcoholism and that alcoholism causes poverty, immorality, ignorance, and atheism.

Reviewing a sixteenth-century classic of the alcohol literature, *On the Horrible Vice of Drunkenness*, written by historian-philosopher Sebastian Franck, E.M. Jellinek illuminated Franck's acceptance of the link between alcoholism and moral insanity in the following words:

> Franck, however, was much more interested in the ethical deterioration of the chronic alcoholic than in bodily ailments. The blunting of emotion, the economic irresponsibility, the untruthfulness, brutality and loss of interest in all the finer aspects of life were the factors which Franck regarded as the greatest perils of habitual inebriety (Jellinek, 1941).

Alcoholism and Moral Insanity — 1985. Recognition of the enormous diversity of alcoholics: rich and poor, white and black, male and female, young and old, bright and dull, moral and immoral, believers and nonbelievers, presidents, kings, professors — and the Skid Row homeless — has become general. Accordingly, no longer do most of us believe, as was the case 50 years ago, that alcoholics come largely from the ranks of the poor, the ignorant, and the morally degenerate.

Vaillant's report (1983) on two 40-year longitudinal studies of normative samples of men, some of whom became alcoholic, is often cited nowadays as evidence of the heterogeneity of the alcoholic. One of Vaillant's samples, originally a control group in the Glueck and Glueck (1950) study of juvenile delinquency, was composed largely of lower socioeconomic status men who had been studied first between 1940 and 1944 while they were students in Boston inner-city schools. As boys, the group had an average IQ of 95; only 33% of their parents had attended high school, 49% of these subjects fell into social classes IV or V (the lowest) as adults. The other group of alcoholic males were originally a portion of a sample of Harvard College students first studied in 1938 (Heath, 1946; Hooten, 1945). In contrast to the Boston inner-city group, 80% of this group came from social classes I and II (the highest), their IQ's ranged between 125–140, and 94% of their parents had attended high school.

Another influential source of data on the heterogeneity of alcoholics in this country comes from the studies of Cahalan and Room (1974). Exploring the nature and frequency of problem drinking among a large, normative sample of men, these investigators concluded that alcohol problems were ubiquitous in all strata of American society. A recent series of papers by Wiens and his colleagues reporting treatment outcome data from a private alcoholism hospital in the Pacific Northwest (Wiens and Menustik, 1983; Wiens et al., 1976) make essentially the same point: Alcoholics in this country are drawn from every walk of life. The data support the contemporary view that alcoholics and prealcoholics are not likely to fit the old stereotypes that portray them as morally insane — as morally, economically, socially, and interpersonally deteriorated. While some may fit the stereotype, so do some nonalcoholics. And while some nonalcoholics function well in the face of serious disease, so do some alcoholics.

Alcoholism and Depression

Alcoholism and Depression — 1935. The psychoanalytic view of alcoholism, extremely influential during the 1930's and 1940's, portrays alcoholism as deriving from strong, unmet dependency needs left over from actual or symbolic loss during early childhood and resulting in strong oral dependency needs in adulthood (e.g., Schilder, 1941). Alcoholism is seen as an effort, one that is ultimately unsuccessful, to recover the lost objects who are, generally, parents. Since depression is also considered by psychoanalytic theorists to be a consequence of early loss, it was not surprising that alcoholism and depression would be seen to share common determinants and that alcoholics would be viewed as commonly depressed.

Alcoholism and Depression — 1985. The decline in popularity of psychoanalytic theories of alcoholism in the past two decades has been paralleled by an increasing recognition that the links between

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alcoholism and depression are reciprocal. Simply stated, it is now recognized that alcoholism often causes depression. It does so because of the enormous personal, vocational/professional, and interpersonal losses the alcoholic suffers from his or her abusive drinking. The pharmacological effects of ethanol also cause depression. It is a sedative drug; only when the body has completely metabolized the ethanol and returned to a stable baseline do the depressive effects of alcohol as a drug disappear. And, reciprocally, depression frequently causes alcoholism when, for example, the depressed individual decides to use alcohol to temper his or her discomfort. In this context, preliminary data supporting a genetic link between antisocial behavior in male relatives, depression in female relatives and alcoholism deserve elaboration (Goodwin, 1985; Winokur et al., 1970).

Alcoholism: A Disorder of Men

Alcoholism: A Disorder of Men — 1935. Fifty years ago, alcoholism was generally felt to be the exclusive province of men. Men were considered more likely than women or youth to demonstrate the antisocial behavior, the immorality and the criminality that were thought typically to accompany alcoholism (and were also assumed to play important roles in its development). As well, alcoholic men came to public attention by seeking treatment in much higher numbers than women. Interestingly, the few women alcoholics who were seen by clinicians were considered even more deviant than alcoholic men and were, as a consequence, more severely condemned than men for their excesses and their deviance. It is little wonder that women alcoholics only revealed themselves as such when they had absolutely no choice about doing so (Bacon, 1945; Dollard, 1945).

Alcoholism: A Disorder of Men — 1985. For at least two decades, alcoholism has been recognized to affect both women and men at high rates. At the same time, it is true that, even in 1985, male alcoholics are more visible than female alcoholics, in part because they are more likely to come to, remain in, and benefit from treatment (Braiker, 1982; Lex, 1985). Most observers have seen this difference in treatment access to be a function of a prevailing orientation, in most treatment facilities, to the values and expectations of males in treatment for alcoholism (Nathan and Skinstad, 1987). Another consequence of these differences in treatment access is a legacy of uncertainty over whether rates of alcoholism for men and women are strictly comparable or only roughly so.

Unhappily, research on the behavioral consequences of alcoholism in women, on women in treatment, and on strategies for preventing alcoholism in women continues to be scarce, for reasons which are not entirely clear. Questions such as whether or not men and women metabolize ethanol at different rates, whether the behavioral consequences of intoxication are the same for men and women at

comparable blood alcohol levels, whether men and women develop alcoholism as a function of the same etiologic factors, whether women and men would benefit best from different or similar treatment programs and therapists, and other crucial issues remain unresolved — and largely unstudied — in 1985 (Nathan and Skinstad, 1987).

ETIOLOGY

Etiology — 1935. For several centuries before, and through the decade of the 1930's, the etiology of alcoholism had been assumed principally to involve a weak will, sinfulness, and a bad moral character. People became alcoholic because, for some unknown, perverse reason, they chose to flaunt society's rules.

With the founding of Alcoholics Anonymous, and as a result of its growing importance in the years thereafter, the disease model, however it was variously interpreted, affected views on alcoholism etiology. Unfortunately, the absence of data to lend flesh to the bones of the disease model made it a target for attack from every quarter, in part because, simply, it was misunderstood. Basically, as I understand the position of AA on etiology, it is that alcoholism is the product of an unidentified, otherwise unspecified, lesion somewhere in the body that is responsible for crucial differences between pre-alcoholics and others in their responses to alcohol. Environment, personality, and psychopathology can all play roles in the form alcoholism takes, but central to every alcoholic is this inborn inability to drink alcohol the way social drinkers consume it.

As I have already noted, it was during the decade of the 1930's that the influence of the psychoanalytic view on alcoholism etiology reached its peak. That view stated that alcoholism is a consequence of excessive deprivation or gratification of dependency needs during the oral stage of infantile psychosexual development. During the decades following, until the early 1970's, the psychoanalytic view was second in impact only to the disease model of alcoholism.

Other etiologic theories saw alcoholism as the burden (and consequence) some of the children of alcoholics have to carry to live with alcoholic parents. It was not clear why living with alcoholic parents increased the likelihood that the child would develop alcoholism, although many believed that the chaos attendant upon a childhood spent with one or two actively alcoholic parents was reason enough to drink abusively as an adult (McPeek, 1944).

Early behavioral clinicians, of whom there were very few in the 1930's, 1940's and 1950's, adopted the uncomplicated view that alcoholic behavior is the means the alcoholic has chosen to deal with conditioned anxiety. This view was supported by two influential

studies by Conger (1951, 1956). Conger reported that when animals were subjected to experimental stress, they consumed substantially more of a solution containing alcohol than when they were not stressed; the alcohol in the solution, presumably, dampened the stress experience. Unfortunately, Conger's "successful" effort to confirm what has come to be called the tension-reduction hypothesis of alcoholism led behaviorists prematurely to extrapolate from data from lower animals to conclude that the theory also has important validity when applied to humans. Many other beliefs about etiology, all unsupported by empirical research, held sway during the decade of the 1930's. Among the most interesting, from the perspective that 50 years provides us, was the widespread conviction that ingestion of distilled spirits was a necessary condition for the development of alcoholism: Beer and wine were believed by many to be incapable of causing addiction to alcohol!

Etiology — 1985. By 1985, the possibility of a genetically-determined predisposition to development of alcoholism has been generally accepted. Operating independently of the environmental impact of living with alcoholic parents (a circumstance that has not yet been linked clearly to the development of alcoholism [Vaillant, 1983]), a genetically-based predisposition to alcoholism was not a central feature of the original disease model of alcoholism that dates back to the late 1930's, despite the support it gives that model.

The most influential of the studies leading to consideration of a genetic predisposition to alcoholism on the part of some children of alcoholics was first reported by Goodwin and his colleagues (Goodwin et al., 1973; Goodwin et al., 1974) and later confirmed by Cloninger and his co-workers (Cloninger et al., 1981). Goodwin found that, in Denmark, adopted male biological offspring of alcoholics raised by nonalcoholics were significantly more likely to develop alcoholism than the biological offspring of nonalcoholics, even though some of the latter were raised by alcoholics. Goodwin and his colleagues (1977) also reported that the role of genetics in predisposing women to develop alcoholism, in contrast, does not appear to be as strong.

Precisely how a predisposition to the development of alcoholism is transmitted is now the object of intense research effort. Best guesses are that it is mediated by the central nervous system. If predisposition to alcoholism is found to involve a difference in central nervous system function between pre-alcoholics and others, either in rate or locus of ethanol metabolism (Li, 1985) or in rate or manner of tolerance development (Nathan, 1982), then a central tenet of the disease model will have been supported.

Undoubtedly processes of learning also play a role in the etiology of alcoholism, but the precise nature of that role remains uncertain. It

is likely that an interaction among learning modes takes place. Simultaneously involved are classical conditioning (by which alcohol consumption comes to be associated with a reduction in anxiety), operant conditioning (by which alcohol consumption is reinforced by peer approval, enhanced relaxation, increased social skill, etc.), and modeling (by which alcohol consumption and its antecedents and consequences are patterned after parents and peers). After learning has taken place, it is then mediated by the drinker's attitudes and expectations surrounding alcohol and alcoholism. Together, the learning and the attitudes and expectations that mediate that learning affect alcohol use and abuse. In other words, it is now clear that the simple tension-reduction model is no longer sufficient, by itself, for an adequate understanding of the development of alcoholism (Marlatt, 1979; Marlatt and Rohsenow, 1980).

Laboratory studies of the social learning mechanisms involved in alcohol consumption by alcoholics and nonalcoholics have demonstrated that learning plays an important role in the behavior of both alcoholics and others (e.g., Bigelow et al., 1972; Mendelson and Mello, 1966; Nathan et al., 1970). However, none of the studies has enabled precise delineation of the nature of the relationship between learning and alcoholism, though many behavioral clinicians believe one exists.

And while behavioral research during the last decade also suggests that cognitive factors play an important mediating role in the development of alcoholism, the precise nature of that role remains to be shown. Unforeseen 50 years ago, the data on the role of cognition in alcoholism suggest strongly that what a heavy drinker or alcoholic believes causes his or her heavy drinking affects the nature and extent of that drinking. And how he or she feels about the likelihood of alcoholism treatment working for him or her plays an equally important role in the success or failure of that treatment (Marlatt and Gordon, 1985; Moos and Billings, 1982).

In like fashion, while it seems clear that psychiatric disorder plays a role in the development of alcoholism, the precise nature of the relationship, if any, is unknown.That is, while some alcoholics drink to excess to dampen anxiety, reduce feelings of depression, or blot out the impact of hallucinations and delusions, it is unclear whether those who suffer from anxiety disorder, major depressive disorder, or schizophrenia, for example, are more inclined to develop alcoholism on either a genetic or a reactive basis (e.g., Barry, 1980; Cloninger et al., 1979; Krueger, 1980).

While a relationship between childhood antisocial behavior and alcoholism has been strongly suggested by the results of both Vaillant's longitudinal research (1983) and Tarter's studies of hyperactiv-

ity in those at risk for alcoholism (Tarter et al., 1984), it is uncertain whether the relationship is causal or simply correlative.

Vaillant's conclusions also suggest that alcoholism is *not* a consequence of inadequate child-rearing practices, emotional turmoil, psychological disorder, emotional deprivation, or poorly- or excessively-gratified dependency needs in childhood. Despite the widespread conviction, during the past 50 years and before, that these phenomena play a causal role in the development of alcoholism (e.g., Cotton, 1979; Donovan, 1986; Zucker and Gomberg, 1986), Vaillant's data suggest otherwise.

TREATMENT

Treatment — 1935. In 1935, bizarre drugs and medicines were still used to treat alcoholism. One was *Mrs. Moffat's Shoo Fly Powders for Drunkenness*, an over-the-counter potion which was condemned in 1935 under the Federal Food, Drug, and Cosmetic Act after a panel of experts testified that it contained enough antimony and potassium tartrate to be classified as a poison. While no deaths following ingestion of the *Shoo Fly Powders* had been reported, it was thought entirely possible that deaths attributed to withdrawal or acute liver failure might instead have been from *Shoo Fly Powder* poisoning. One troubling aspect of the case was that, for approximately 60 years, the manufacturer of Mrs. Moffat's had sold about 50,000 powders annually to persons prepared to testify that it was an effective treatment for drunkenness.

To this end, it is worth noting, as well, that for centuries ethanol itself was considered a valuable therapeutic agent. A note in a 1930 popular journal, for example, observed that while per capita consumption of alcohol in hospitals had declined an average of 40% between 1906 and 1922, the U.S. was still reporting a consumption level of seven liters of alcohol per hospital patient per year, all for "therapeutic purposes." In this context, it is interesting to observe that per capita consumption of alcohol in the U.S. in 1981 was 2.8 gallons (NIAAA, 1983); seven liters constitutes a little more than half this total.

It seems likely that detoxification from alcohol was more common in jails than in hospitals or clinics 50 years ago, since public drunkenness was a crime in most places until the 1970's, and since persons were also jailed for other crimes committed while they were intoxicated. It is also certain, in the same context, that a number of deaths occurred in jails from complications of alcohol withdrawal.

Inpatient alcoholism treatment and rehabilitation were less available 50 years ago than they are now. Moreover, inpatient treatment, when it was offered, was housed more often in state mental hospitals and prisons than in facilities devoted exclusively to alcoholism treatment.

At the same time, influential advocates of dedicated treatment programs did exist. For example, famed Johns Hopkins psychiatrist Adolf Meyer said: "the most dependable means available (for alcoholism treatment) are asylums for drunkards with more or less efficient provisions for after care, insisting on total abstinence, during the period of physical and character reconstruction" (Meyer, 1932). Meyer's thinking accords well with current theories of treatment in most regards, except his view that the alcoholic's character must be reconstructed during treatment to bring his moral sense and character back up to normal levels.

Despite Meyer's convictions, however, there were only a few private hospitals for the treatment of alcoholism, and only a few private psychiatric hospitals with wards for alcoholics; there were even fewer public facilities specifically designed for alcoholism treatment. Hence, the alcoholic who both needed and wanted a period of inpatient treatment but did not have the means to pay (there was little third-party reimbursment during the 1930's) had to seek it in inadequately-funded state mental hospitals. Treatment in such places was typically superficial and laden with moral imperatives; the stigma of alcoholism in the state hospitals was as profound as the stigma of tertiary neurosyphilis, still a source of many patients in those hospitals during that time.

In the decade of the 1930's, involvement in alcoholism treatment by professionals, especially physicians, was minimal. Doctors saw alcoholics when they were called on to treat the long-term physical effects of alcoholism (which they recognized as such only some of the time). And when alcoholics did suffer from medical complications, they were limited in their access to general hospitals because general hospitals resisted admitting known alcoholics, even when they were suffering from life-threatening conditions. That resistance, unfortunately, continues in muted form to this day.

A strongly judgmental, moralistic, condemnatory, infantilizing view of the alcoholic in treatment prevailed in the 1930's and for centuries before and decades after. In 1934, for example, Professor Yandell Henderson of the Yale Center of Alcohol Studies was said to have observed that "A man of strong will can reform from chronic inebriety more easily than a dipsomaniac or morphinist," suggesting that strong will was required for recovery just as weak will had been essential to initial descent to alcohol addiction.

Prior to 1935 and the founding of Alcoholics Anonymous, there appears to have been little in the way of a tradition of self-help for alcohol problems. And, of course, it took several years before the Fellowship began to grow and groups began to proliferate nationwide. The slow growth was probably because of the stigma attached to alcoholism 50 years ago, a reason to admire enormously the

courage of the founders of AA for being willing to identify themselves as alcoholics in the face of the prejudice they knew they would experience even as anonymous founders of the Fellowship.

There also appears to have been uneven recognition, until very recently, of alcoholism as a chronic, recurrent, lapsing disease. The founding of Alcoholics Anonymous and its emphasis on lifelong sobriety and, much later, the cognitive social-learning theorists' emphasis on relapse as a cognitively-mediated phenomenon have contributed to this recent important discovery.

There appears to have been little or no recognition 50 years ago of the possibility that treatment programs designed for white, middle-aged, male alcoholics might not be equally helpful for women, non-Caucasians, the young and the old. That discovery, in fact, has been only a very recent one. A fascinating overview of early efforts to treat alcoholics was published recently by Mark Keller, longtime editor of the *Journal of Studies on Alcohol* (Keller, 1986).

Treatment — 1985. A plethora of treatment approaches, personnel, facilities, and beliefs had developed by 1985. Self-help groups like AA, Al-Anon, and Alateen have become very visible and influential. Out- and inpatient treatment, halfway houses, and night and day hospital programs all claim their adherents. Some alcoholism workers believe that drugs and hospital settings work best while others advocate nonmedical detoxification and treatment. Some workers believe that professionals are most effective with alcoholics while others believe as strongly that recovering alcoholic paraprofessionals work best. Treatments that focus on the individual and his or her pathology, the family and its pathology, the stress of the worksite and the community, all have been developed and all have their supporters. Treatments based on behavioral, cognitive, social-learning, psychoanalytic, family systems, rational emotive, multimodal, client-centered, and primal perspectives are offered. In other words, from a paucity of treatment programs and approaches in 1935, an overwhelming array of different programs and approaches have now developed.

Today, alcoholism treatment, irrespective of content or theoretical orientation, rarely focuses on moral reeducation. In like fashion, alcoholism workers seem much less convinced that their patients are morally deficient. In fact, there are considerable data that now suggest that alcoholics do not differ along most dimensions, including moral sense, from nonalcoholics from comparable cultural and socioeconomic backgrounds (Cahalan, 1982; Vaillant, 1983).

There is greater recognition of alcoholism as a chronic disease. This change, which affects treatment programming in useful ways, represents an important, unintentional collaboration of Alcoholics

Anonymous (which has emphasized both the chronic and the recovery aspects of alcoholism) and the behavioral social-learning perspective. The latter view, epitomized by Marlatt and Gordon (1985), focuses attention on the phenomena of the recovery period, including the need to understand episodes of relapse.

Self-help groups are now centrally involved in alcoholism treatment, both because a greater variety of treatment is being undertaken than ever before and because many have become convinced that self-help may be the best chance for recovery from alcoholism. The self-help groups are mostly Alcoholics Anonymous or allied groups; AA has become a treatment of choice for many alcoholics and almost certainly the most widely known and well-respected treatment modality worldwide.

Professionals are also much more involved in alcoholism treatment, reflecting to an equal degree the lessening of the stigma attached to working with alcoholics, the increased scientific respectability accorded research on alcoholism, and the increased availability of third-party reimbursement for the treatment of alcoholics by professionals.

Both because public drunkenness is no longer a crime in most places and because of the negative consequences of unattended detoxification in jails, detoxification is now accorded more concern by trained alcoholism workers. Alcoholics are now more often detoxified in hospitals and nonmedical facilities than in jails. As a result, significantly fewer persons die of complications of withdrawal now than did 50 years ago.

We now recognize, in the decade of the 1980's, that rates of recovery from alcoholism vary with certain prognostic signs, most of them specific to the individual, including age, vocational status, marital status, level of motivation for change, prior drinking history, and prior treatment experience. By contrast, rates of recovery seem to depend much less on factors specific to treatment, like content, form, theoretical orientation, or intensity of treatment (Emrick, 1974, 1975; Nathan & Skinstad, 1987). In other words, few consistent differences in outcome have been reported for one or another of the multitude of different treatment programs, methods, and procedures developed for the treatment of alcoholism during the recent decades. The data on individual predictors of treatment outcome, in turn, have led to concerted efforts to determine the feasibility of matching treatment to the individual most likely to benefit from it (Moos and Billings, 1982).

These data on predictors of treatment outcome, most gathered during the 1970's and 1980's, have brought into question a belief that characterized the 1930's and intervening decades, to the effect that

there is a single treatment method more successful than any other. For a time, the treatment of choice was individual psychoanalysis, for a longer time it was intensive involvement in the fellowship of Alcoholics Anonymous, for a time behavioral approaches to treatment were considered most promising. Yet current data on predictors and factors in treatment outcome indicate that there is no single approach which yields more positive outcome figures than any other, despite the belief on the part of many even now that there is such an approach (Armor et al., 1978; Polich et al., 1981; Vaillant, 1983).

Another long-maintained belief about treatment still held by many despite the absence of empirical support is the conviction that recovering alcoholics are better able to understand and treat other alcoholics than those who have not themselves experienced alcoholism. This belief persists, like the belief that one treatment method is superior to all others, in the face of data to the contrary, data which indicate that therapist training, background, and identity (as recovering alcoholic or not) do not exert a demonstrable impact on treatment outcome (Emerick, 1974, 1975; Nathan and Skinstad, 1987; Armor et al., 1978; Polich et al., 1981).

PREVENTION

Prevention — 1935. Two principal prevention approaches characterized the 1930's. Though Prohibition extended only through the early 1930's, its numerous supporters continued to lobby for limited prohibition, to the extent possible, after repeal of the Eighteenth Amendment. But there were also those who recognized that Prohibition, however desirable its goals may have been, was simply unworkable in our society. In its stead, they proposed making the sale and distribution of alcoholic beverages as difficult as possible, by imposing taxes that would markedly raise the price of alcoholic beverages, erecting legal restraints on sales to certain groups of people (e.g., those below a certain age), and restricting sales and distribution to certain places and certain hours. Despite strong support by many people, there continues to be active debate on whether such restrictions on availability affect the prevalence of alcoholism and alcohol-related injury or death (Nathan, 1983).

The decade of the 1930's and before also witnessed advocacy for a different — and distinctly unproven — control strategy: Restricting the sale of high-proof beverages, in the mistaken belief that distilled spirits are largely or solely responsible for alcoholism. Thus:

> "From the point of view of the prevention of drunkenness, the superiority of the more dilute beverages, such as the lighter beers and natural wines, is therefore mainly due to the fact that the bulk of the fluid makes it very difficult for the drinker to consume a very large dose of alcohol within a moderate period" (Report of the Senate Judiciary Committee on Modification of the National Prohibition Act, quoted by Henderson, 1934).

"If we could largely confine our drinking to beverages below 15 or 18 percent of alcohol by volume, the peculiar American problem would largely disappear, alike in its individual, its social, and its political aspects. Since 3.2 beer was declared legally nonintoxicating in March, 1933, and prior to the reopening of the saloons after repeal of prohibition in December of that year, light beer has proved to be an effective agency to replace and diminish to an appreciable degree the consumption of spirits. The drinking of beer by young men, particularly college students — those under as well as those over twenty-one — should be encouraged as a means of keeping them away from spirits" (Henderson, 1934).

Restrictions on the advertising of alcoholic beverages were also proposed in 1935, in the belief that this would decrease rates of alcoholism. Billboard advertising of alcoholic beverages was targeted for legislative restriction in 1935 and, in 1939, proposals before the U.S. Congress would have prohibited advertisements on radio.

Another prevention effort characterizing the 1930's (and for centuries before and decades after) involved trying to make alcoholics an example to others who might be tempted to become alcoholic, by treating them punitively and judging them harshly. Fines for public drunkenness, for example, were widely imposed although they varied enormously in amount. In 1939, for example, fines for public drunkenness ranged from 50 cents an occasion in Delaware and $1.00 in New Jersey to $3,000 and two years in jail for a second offense in Florida! No data exist to support the view that fines for public drunkenness altered rates of alcoholism anywhere.

Prevention — 1985. Alcohol prevention strategies in the 1980's have concentrated on education programs for high-risk and special groups, including adolescents, women, the elderly, and minorities. These programs generally have two immediate aims: first, to educate on the behavioral, psychological and psychiatric, and physiological actions of alcohol, on the range and variability of both normal and abusive drinking in the United States, and on alcohol's short- and longer-term effects in both social and abusive drinkers. The second aim is to attempt to change attitudes toward consumption, especially heavy consumption, so that those attitudes will become more negative — or at least more wary. The ultimate aim is to reduce overall consumption levels and, especially, incidence and prevalence of abusive and alcoholic drinking.

Other current prevention efforts harken back to the decade of the 1930's and before. These include legal efforts to restrict the availability of alcoholic beverages by raising the age for purchase and consumption, increasing taxes, demanding that labels warning of the health consequences be put on alcoholic beverages, and advertising restrictions to decrease demand.

This decade has also seen the imposition of strong legal and professional efforts to diminish certain consequences of alcoholism most damaging to innocent members of society. These efforts include an attack on drunken driving, with more stringent enforcement of drunk driving laws, random highway checks of blood alcohol level, and enactment of federal legislation to encourage all states to set the legal age to purchase alcoholic beverages at 21. In response to the fetal alcohol syndrome, federal and state programs have been funded to encourage pregnant women and women of child-bearing age to reduce or stop drinking, train physicians and nurses to identify pregnant women who are at risk, and alert the general public to the hazards of drinking alcohol during pregnancy (Nathan, 1983; NIAAA, 1983, 1987).

The net result of current efforts to prevent alcoholism has been disappointing. These efforts have consistently yielded reports of increased public awareness of the hazards of heavy alcohol consumption and of desirable changes in attitudes towards drunkenness and alcoholism, yet few instances of documented change in levels or patterns of alcohol consumption as a result of alcohol education have been reported (Nathan, 1983).

By contrast, efforts to diminish the incidence and prevalence of two specific consequences of abusive alcohol consumption — drunken driving (especially by teenagers) and the fetal alcohol syndrome — have been been positive. Both rates of drunken driving by teenagers and incidence of fetal alcohol syndrome have decreased while arrests for drunken driving have increased in recent years (NIAAA, 1983, 1987).

CONCLUSIONS

1. Significant progress has been made during the past 50 years in identifying and understanding important effects of alcohol on human behavior. The dimensions of behavioral pathology induced by acute and chronic alcohol abuse have been much better delineated. Behavioral scientists have played important roles in this successful search for understanding.

2. Significant progress has also been made in demonstrating the parameters of the genetic predisposition to alcoholism that seem to characterize some of those with at least a single alcoholic parent. However, the extent of the impact of a genetically-based predisposition to alcoholism, the proportion of those with an alcoholic parent who carry this predisposition, the factors governing the expression of the predisposition, or the manner in which the predisposition is

transmitted, remain uncertain. Behavioral scientists are involved in a major way in studying the latter issue.

3. Progress in research on other factors involved in the etiology of alcoholism, however, has been limited by the expense, complexity, and difficulty of longitudinal research. The best strategy is still to be determined for studying the psychosocial, environmental, and learning-based factors involved in the natural history of the disorder. As a consequence, much less progress has been made in identifying these particular etiologic factors of special interest to behavioral scientists.

4. In contrast to progress made in identifying the behavioral pathology and etiologic factors associated with alcoholism, relatively less progress has been made to develop effective treatment programs. In fact, it now seems unclear how much treatment variables *per se* add to the variance associated with the decision by an alcoholic to stop drinking. More important than treatment variables, which include form, locus, and intensity of treatment, are patient variables: the alcoholic patient's marital and vocational status, his or her age, sex and education, and, above all, his or her motivation to change the pattern of drinking.

5. The progress in the treatment area that has been made in the past 50 years has involved the development of research strategies to reach reliable outcome assessments and identify predictors of outcome. The elimination of "moral re-education" approaches to treatment probably ought to be considered progress as well.

6. Only modest results have been achieved in the prevention area in the past decades, despite significant expenditures of federal and state funds. A complete rethinking of strategy and tactics is clearly called for in this area, whose major successes have been in the reduction in the consequences of alcohol abuse rather than in abusive consumption itself.

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About the Center of Alcohol Studies

The Center of Alcohol Studies was founded at Yale University in 1940. The center has been a leader in the interdisciplinary research on alcohol use and its effects and has been in the forefront of the movement to recognize alcoholism as a major public health problem. Dr. E.M. Jellinek was the center's first director, and the prestigious *Journal of Studies on Alcohol*, still published by the center, was founded by Howard W. Haggard, M.D. In 1962, the Center of Alcohol Studies moved to Rutgers University.

The center faculty have been trained in biochemistry, economics, physiology, psychology, psychiatry, sociology, political science, public health, education, statistics and information science. The faculty teach undergraduate, graduate and continuing education courses, including the world famous Summer School of Alcohol Studies. The SSAS alumni have assumed leadership positions in research, prevention and treatment of alcohol problems.

The center's four major areas of concern are: research, education, treatment and prevention. As part of the center's educational mission, this pamphlet series presents information on important topics in the alcohol studies field.